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SPONTANEOUS CURE OF RETINAL DETACHMENT.

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In July, 1885, a farmer, 74 years old, was brought to my office by his brother to see what I could do for his right eye. As far as he remembered he could never see much with the left eye: he had to depend entirely on his right eye which though always very near-sighted, enabled him to do his farm work until two months ago when the sight rapidly failed. He could then see the motions of my hand only in front and in the lower portion of the field; in the upper field there was no perception at all. Tension decidedly diminished, lens clear and vitreous free from opacities; the lower portion of the retina detached, gray and undulating with every movement of the eyeball.

The left eye exhibited a mature senile cataract; normal tension, good perception and perfect projection in every portion of the visual field; in short everything indicated a normal condition of its fundus and warranted a favorable prognosis for an operation except the patient's statement that he never could see much with this eye. But even assuming a certain degree

of congenital amblyopia to exist, I believed the patient had a better chance of recovering a useful degree of sight by an operation upon the cataract of the left eye than by any treatment of the right eye, in view of the fact that a retinal detachment in a highly myopic eye is generally admitted to be a pretty hopeless case.

I, therefore, proposed to leave the right eye alone and to operate upon the left eye giving the patient a full explanation of my reasons. He consented, and on July 24 the cataract was successfully removed; the patient being able to count fingers directly after the operation. Both eyes were bandaged, and the patient spent the day sitting in a rocking chair. The recovery was very rapid; after 10 days the patient could be discharged. But picture my surprise when on examining the right eye I found no trace of retinal detachment; the fundus absolutely normal in appearance, except a narrow crescent (conus); and V=$^{20}/_{LXX}$ with—4. One year later, in September, 1886, I had an opportunity to examine this case again; the detachment had not returned; sight had improved to $^{20}/_{L}$, and the re-attached lower portion of the retina had recovered its perception as shown by the measurements of the visual field extending upwards to 40°. And the same status was found at another examination made Oct. 10, 1887.

In the XI Vol. of the *Archives of Ophthalmology*, Prof. Schweigger reported four similar cases in which a detachment of the retina seen by himself at one time had disappeared at a later period though no treatment whatever had been applied, the patients not even changing their habits and mode of living. Such cases of spontaneous recovery from extensive detachment of the retina are not common, but there is no doubt of their occurring occasionally; and it is advisable to keep this occurrence in mind when one is experimenting with a new treatment for this disease, lest he will too readily attribute to his treatment an occasional cure which in reality has been a case of spontaneous recovery.

Such mistakes in logic are committed and published every day. Suppose a surgeon has employed the same medical or

surgical treatment in 10 cases of detachment, and the result was one cure and nine failures; is it sound logic to attribute this one success to the mode of treatment? or is it not more reasonable to consider the 9 failures sufficient evidence of the insufficiency of the therapeutical efforts? Can we sanely believe in the efficacy of a remedy which fails nine times out of ten? Does an exception prove more than the rule?

In the eyes of some oculists, it seems, it does, or they would not think of recommending procedures against detached retinæ for which they have to show very few good results only, but a long list of failures. The last International Congress furnished a very good illustration on this point, when Dr. Galezowski dilated upon the merits of treating detachment of the retina by aspiration of the fluid under the retina. According to this statement he cured 4 cases out of 25, but he forgot to say whether these "cures" lasted one week or one year; nor did he show why the aspiration of the subretinal fluid should be more efficacious and successful than the withdrawal of this fluid by tapping or by the hypodermic syringe; for there is certainly no radical difference between aspiration and these latter procedures, either of which has accomplished some cures; but most of them proved to be so ephemeral that the most ophthalmic surgeons, I believe have lost faith in these operations. And when during the discussion one gentleman pronounced Dr. Galezowski's procedure "a real addition to the rational means at our disposal; a tremor of surprise must have run through the audience; and it is to be regretted that the gentleman was not requested to name some of those "rational means at our disposal;" for among all those I know there is none which deserves this designation in the light of our present knowledge of the pathology of retinal detachment.

The researches of Iwonoff, Leber, Nordenson and others have clearly shown that the detachment of the retina is preceded and induced by a shrinkage of the vitreous humor. As the sclera is too rigid to adapt itself to the reduced bulk of the vitreous humor this body in shrinking will either detach itself from the retina (detachment of the vitreous,) or the latter will

follow the receding vitreous and become separated from the choroid, if the connection of the retina with the vitreous is firmer than that between the retina and choroid. The space which separates the detached retina from the choroid must, of course, be filled by something; and thus it is obvious that the subretinal fluid is but the necessary physical consequence of the primary pathological shrinkage of the vitreous. But if this view is correct we must regard as irrational every therapeutical effort which is directed against the subretinal fluid for the purpose of curing a detachment of the retina. Unless we can stay the skrinkage of the vitreous the removal of the subretinal fluid cannot be of any lasting benefit; and could we remove that which makes the vitreous shrink, could we restore the normal nutrition of the vitreous so that it would recover its normal size, the subretinal fluid would most likely be absorbed under the increasing pressure by the vitreous, and thus the retina would become re-attached without any operative interference. And I am firmly convinced the successful treatment (if ever we shall find one) of retinal detachment will never be an operation directly attacking the detached retina and the subretinal fluid.

A CASE OF INSUFFICIENCY OF THE INTERNI IN WHICH AN EXTERNUS WAS CUT BY DR. C. R. AGNEW.

REPORTED BY DR. D. WEBSTER, NEW YOEK.

Arthur H., æt. 17, came under observation on Sept. 28, 1874. He had been, for some time, studying hard, preparing for Yale College. His eyes had been weak for two or three years, but gave out entirely only a few days before we saw him. He had been in the habit the preceding winter of rising at 4 o'clock and studying by bad gaslight.

Upon testing his eyes we found that each had vision $^{20}/_{xx}$, and was apparently emmetropic.

Ophthalmoscopically we found no lesion, physiological excavation of both optic disks and apparent emmetropia.

Tested with prisms and candle he showed insufficiency of his interni 12° at 20′, 21° at 1′.

On approaching an object of fixation to the eyes, the left internus would give way, and the left eye would slowly turn out.

Feb. 2, 1875. Insufficiency of interni 12° at 20′, 15° at 1′. V. $=^{20}/_{xx}$. E. both. Ordered atropia.

Feb. 3. After four instillations of a 4 grain solution of sulphate of atropia extending over sixteen hours, V. $=^{20}/_{xx}$, each without a glass. The patient reads all of $^{20}/_{xx}$ *less clearly* with $+^{1}/_{72}$, $+^{1}/_{60}$, or $+^{1}/_{48}$. Glasses $+^{1}/_{48}$ were now ordered for all near work.

April 10. Insufficiency of interni 12° at 20′, 12° at 1′.

Feb. 21, 1877. Insufficiency of interni 13° at 20′, 15° at 1′.

March 9. Insufficiency of interni from 11° to 16° at 20′, 15° at 1′.

March 10. Tenotomy of left externus under ether.

March 13. No insufficiency. The patient was troubled with diplopia for a short time the next morning after the operation.

Mar 14. Insufficiency of interni 1° at 20′, 0° at 1′.

March 22. No insufficiency for near or for far.

April 4. Insufficiency of interni 12° at 20′, 8° at 1′.

April 6. Insufficiency of interni 9° at 20′, 4° at 1′.

The patient states that the second week after the operation his eyes felt well and he could read the newspapers without discomfort. For the last week they have been getting steadily worse. They "make him stupid" after reading a short time, or on exposure to gaslight.

The patient was now put upon reading tasks by Dyer's method.

April 11. Insufficiency of interni 8° at 1′, 8° at 20.′.

There are no further notes in this case, but I am under the impression that the patient was only partially relieved, as the insufficiency was only partially corrected.

So high a degree of insufficiency is uncommon, I think, in eyes that are emmetropic.

DETACHMENT OF THE RETINA IN THREE SUCCESSIVE GENERATIONS OF ONE FAMILY.

BY ADOLF ALT, M.D.

On the 11th of July, 1887, A. H., 49 years of age, consulted me about his right eye. His statement was, that during the week preceding this visit he had gradually lost the sight of the right eye without his knowing any possible cause for it.

I found vision reduced to fingers at two feet outwards and upward; straight forward to perception of light. —T. The visual field was reduced to a small area up and outward. The retina could easily be seen floating in the muddy vitreous. The left eye was found to be emmetropic and presbyopic.

The patient stated that his mother had lost an eye in a similar way, and, furthermore, that his right eye had always been weaker (probably myopic.)

Rigid rest of eye and body, injections of pilocarpine and sublimate internally seemed for a time to bring about an improvement. The visual field became considerably larger and he counted fingers at seven feet 2 weeks after the first examination. Yet, as soon as he sat up for even a short time, the former condition returned, and considering the case incurable I discharged the patient. His eye has since remained the same.

On the 19th of September last, I was consulted by Mrs. N. L., 18 years of age, the daughter of A. H., on account of sudden loss of vision in her right eye the day before. Standing on a chair she had fed a canary bird, and when stepping down she got suddenly very dizzy and blind in the right eye. She had been married two months and was pregnant. She was always myopic and wore glasses.

I found vision reduced to $^{2}/_{cc}$ excentrically. —T. Visual field upward, outward and inward wanting. The ophthalmoscope showed a corresponding detachment of the retina, which latter was, however, not far enough detached to be floating.

Rigid rest of eyes and body, injections of pilocarpine and hydrargyrum tannicum internally at once brought about an improvement of the conditions. Six days after the first consultation, the visual field had almost the normal area. The retina was everywhere reapplied to its natural support and although a little shadow is yet complained of upward and outward the ophthalmoscope does no longer show anything abnormal.

Detachment of the retina having thus occurred in the father and daughter, I became doubly interested in knowing exactly what disease had rendered Mrs. N. L.'s grandmother blind.

The following portion of a letter by Dr. G. Strube, of Bremen, Germany, made it certain, that she also had suffered from detachment of the retina. The doctor writes: "I saw Mrs. H. in 1858. She had then been in Berlin and under the treatment of von Graefe. According to his statement there was detachment of the retina in a myopic eye. Iritis and total posterior synechia had supervened and von Graefe had made an iridectomy. After this the inflammatory symptoms had ceased. The eye remained unchanged, except for the development of a cataracta accreta, until her death which occurred about ten year later."

Thus undoubtedly mother, son and grand-daughter in direct succession became subject to detachment of the retina.

THE BOWMAN LECTURE ON THE VALUE OF EYE SYMPTOMS IN THE LOCALIZATION OF CEREBRAL DISEASE.

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Delivered before the Ophthalmological Society, Nov. 9, 1888.

MR. PRESIDENT AND GENTLEMEN.—Recent advances in cerebral surgery have brought with them, in some respects, increased responsibility, and increased anxiety for the surgeon, in cases of focal cerebral disease. In these cases perhaps the gravest responsibility and anxiety is encountered at the very outset, when the regional diagnosis has to be made. It is true that our present knowledge of the anatomy, physiology, and pathology of the brain frequently enables us to diagnose the position of focal lesions; but it must be confessed there remains much to be learned before we shall be able to say, that in all of the cases met with localization can be successfully effected. Yet we may confidently hope that the difficulties which still beset this subject will gradually disappear; and we are encouraged to think so by contemplation of the marvellous progress which has been made in cerebral physiology within the last eighteen years.

In order that the desired end may the sooner be attained, it is of the utmost importance that the symptoms of each case of focal cerebral disease should be carefully noted, and that each necropsy should be conducted with the greatest precision; and in looking over the records of such cases, one cannot help observing that it is just in respect of the necropsy that most of them are defective. I do not mean merely that

in many of the cases which ended fatally no necropsy was, or probably could be, made, but also that in many instances where a post-mortem examination was obtained the record of it affords only scant or inaccurate information as to the seat and extent of the disease.

I feel sure we shall see quicker and better progress in cerebral localisation when many physicians and surgeons recognize more fully than they yet have done, that the interests of science, as well as their own reputations, are best served by entrusting the examination of the brain in all these cases to skilled pathologists and microscopists, rather than by undertaking the necropsies themselves; for it must, I think, be admitted that the successful examination of a diseased brain requires special training. The mere fact that marked focal symptoms may be caused by minute cerebral lesions is sufficient to demonstrate the importance of this view, without dwelling upon it further.

In the meantime, while our knowledge of the physiology and pathology of the brain is gradually increasing, we must be careful in our daily practice to employ all the facts which have hitherto been acquired concerning this interesting organ; and it has occurred to me that it might serve a useful purpose were I to occupy the time at my disposal this evening in reviewing the symptoms which are derivable from the eye in cases of focal cerebral disease, and in considering how far they may be utilized for the localization of cerebral lesions. I incline to the opinion that these eye symptoms are not as much valued as they should be, perhaps because their often subtle and sometimes subjective nature renders them less readily studied than are other focal brain symptoms.

I also incline to think that eye symptoms are too often not looked for at first, but utilized rather as a *dernier ressort.* This is a serious error, for many of these symptoms are of the highest localising value; while their very nature may render their discovery in a late stage of the disease impossible. How, for example, can a field of vision be examined in a late stage of tumor of the brain when the patient is racked with pain, delirious or half unconscious?

And if it be remembered what a prominent *rôle* in cerebral regional diagnosis the process of exclusion must necessarily play, and what a very large extent of the brain is placed in relation to the eye, it will be admitted, that when a search for eye symptoms in a case of focal brain disease gives only a negative result, yet the significance of this for the diagnosis falls little short of that which belongs to a positive result.

In endeavoring to accomplish the task I have set before me, I invite your attention to the facts provided for us by clinical pathology, rather than to those of experimental comparative physiology. For, important as are experiments upon animals for our knowledge of the physiology of the human brain, yet no one will deny that the evidence of the bedside and of the post-mortem table, when it can be obtained, is of even greater importance. You will understand, then, why it is that I refer but little in the following to the epoch-making researches of such distinguished men as Ferrier, Schaefer, Victor Horsley, Hitzig, Munk, Flechsig, Goltz, and many others.

Gentlemen, in the details of this lecture I can hardly hope to offer anything that is new to you, yet I trust that in this respect you will extend to me your indulgence, and that you may be able to find at least some interest in the presentation of the subject as a whole.

In estimating the localising value of the focal eye symptoms which may be afforded by a given case of recent brain disease we are immediately confronted with a difficulty which is common to all focal symptoms, the difficulty, namely, of distinguishing between direct symptoms and the so-called indirect symptoms; the former, as you are aware, being those which depend upon the loss of function of the part in which the lesion is situated, and which, consequently, are the symptoms it is desirable to point out, while the indirect symptoms are not the result of the local disorganization caused by the lesion, but of its pressure, of disturbances of circulation to which it gives rise, and, it is also thought, of inhibition effects, all of these being liable to interfere with the function of parts of the brain more or less distant from the lesion.

I agree with Dr. Gowers in thinking that the term "indirect" conveys a false conception of the nature of the symptoms to which it is applied, and I venture to suggest to you in its place the term "distant symptom" as being more suitable. It is an adaptation of the *Fernwirkung* of some German writers, and implies, I think, the one fact of which we are certain in connection with these symptoms, and no more.

We may at once say that there is nothing in the manner in which focal eye symptoms present themselves which can enable us to distinguish between the direct and distant symptoms. It is only in cases of stationary lesions, which remain under observation sufficiently long—some six weeks or more —after the first onset, that we can feel pretty sure that the symptoms before us are direct symptoms; for, after that interval, it is probable that the distant symptoms, if any have been present, will have passed away. But, in the case of a tumor, which constantly and slowly increases in size, distant symptoms may come and go, and then we can often only recognize the direct symptoms by their greater constancy. No doubt some symptoms are more often distant than others, and it will be my duty, as I go along, to endeavor to point out those which seem to be, respectively, the most and the least prone to be distant.

The opposite of those cases, in which not only direct but also distant focal symptoms are present, is afforded by cases where the lesion is latent, where it gives rise to no focal symptoms whatever. There is probably no part of the brain in which a destructive lesion may not be present without producing any focal symptoms, and this is one of the most remarkable facts connected with cerebral disease. Here the presence of diffuse symptoms renders the existence of focal brain disease indubitable, while the absence of focal symptoms renders localisation of the disease impossible. Lesions in those parts of the brain which, as a rule, give rise to eye symptoms, may sometimes be latent, like lesions elsewhere. Marked disease, for example, in the occipital lobe, in the internal capsule, in the cerebral peduncle, and so on, may fail to produce the

accustomed eye symptoms, or any others. It was necessary that I should refer to this interesting point, but its further consideration does not come within the scope of this lecture.

Focal eye symptoms may be divided naturally into those which depend upon disturbances in the motor apparatus of the eyeball, including the intraocular muscles, and those which depend upon disturbances in the special visual apparatus. We have also to consider symptoms due to lesion of the nerve of ordinary sensation of the surface of the eyeball.

And, first, as regards the symptoms derivable from the motor apparatus. In the cerebral cortex, centres exist for the motions of the face, arm and leg, respectively, but no centre, in precisely the same sense, for the motions of the eyeball is present; that is to say, there is no centre in the cortex of one hemisphere a lesion of which will produce ophthalmoplegia, partial or complete, of the opposite eyeball alone, although attempts have been made to describe some such centres. All are agreed that the reason for this is, that the two eyes being associated in their motions, it is only those associated motions which are represented in the cortex.

By far the most common derangement of these associated ocular movements, as the result of cortical lesions, is the conjugate lateral deviation of the eyes to one side—that is, abnormal function of the internal rectus of the one eye, and of the external rectus of the other eye. In paralysis, the deviation being, of course, toward the side of the lesion, the eyes look at the cerebral lesion, as Prevost has expressed it; and in spasm, from the side of the lesion. In many of these cases, if the patient's intelligence be not impaired, the eyes can be moved as far as the middle line, or even further by an effort of the will. We are not able, as yet, to say where the cortical centre for these motions resides in man, for Grasset's statement[1] that it is situated in the supra-marginal and angular gyri has not been verified.

But, did we know its position, it is not likely that much

[1] De le Deviation Conjugée, Paris, 1879.

would be gained, so far as clinical localisation is concerned, for this centre seems to be extremely sensitive, and to be easily thrown out of gear by lesions of many different parts of the cortex. Conjugate deviation is, in short, very apt to be a distant symptom, especially in cerebral hæmorrhage, when it is often accompanied by a rotation of the head in a corresponding direction, and lasts only a brief time. Moreover, it is thought that, when this centre may happen to be actually involved in the lesion, its function, being largely bilateral, is rapidly taken up by the opposite hemisphere; and hence, even when conjugate lateral deviation plays the part of a direct cortical symptom, it can never be recognized as such, owing to its evanescent character.

Again, conjugate lateral deviation similar in character to that caused by a cortical lesion may proceed from a lesion of the internal capsule.

This same symptom, finally, may be caused by a lesion of the pons, involving the special nucleus for the associated motion, which is common to the sixth and third nerves, although distinct from their proper nuclei, and which probably resides in the superior olivary body. But conjugate deviation from a lesion in this locality differs from the same symptom in a case of lesion of the cortex, or of the internal capsule, inasmuch as the eyes are here turned away from the side of the lesion, toward the hemiplegic side of the body, in paralysis, and toward the side of the lesion, away from the convulsed side of the body, in irritating lesions. Gowers, in his recent exhaustive work on "Diseases of the Brain,[1]" points out that in these pontine lesions the symptoms vary according as the disease does, or does not involve the nucleus of the sixth nerve. In the first place, if the disease is above the nucleus of the sixth nerve, that is, at or in the neighborhood of the superior olivary body, the eyes cannot be moved toward the side of the lesion beyond the middle line, but in some cases, although the associated movement is lost, yet convergence can

[1]Page 167.

be effected. But, if the very nucleus of the sixth nerve be involved, there is complete loss of power of the external rectus, so that the eye deviates inward, and cannot be rolled outward, while the other eye can be moved by its internal rectus as far as the middle line and no further. Attention was first drawn to this form, I think, by Broadbent.[1] In it the facial nerve, on the side of the paralyzed sixth nerve, is often also paralyzed, owing to the proximity of its fibres to the nucleus of the sixth nerve; or because the sixth and portio dura have a common nucleus. Finally, if the fibres of the sixth nerve in the pons, after they have left their nucleus, be injured, the external rectus is paralyzed, but the associated internal rectus of the other eye is not impaired in its function. Conjugate deviations due to pontine lesions differ again from those due to cortical or capsular lesions, in that they are almost always direct symptoms. Doubtless the reason for this is, that the close proximity of the two nuclear centres for the associated lateral motions of the eyes to each side, respectively, lie so close together that a distant effect can hardly be produced upon one of them alone. Yet if both nuclear centres should happen to be paralyzed, we must not at once conclude that the resulting symptom is a distant one, for, according to Wernicke,[2] cases of simultaneous focal lesion of both of these centres have been observed. The clinical appearance is then very peculiar, the eyes being directed straight forward, and being absolutely immovable to the right or to the left, while their upward and downward motions are preserved, as well as the motions of the upper eyelids.

Conjugate lateral deviation of the eyes, then, may assist us in the diagnosis of a cortical or of a capsular lesion from one in the pons. It may aid us in deciding in which side of the brain a lesion is situated when other symptoms are not readily observed, as in coma. And the varieties of the symptom, when it is due to disease in the pons, may enable us to form a refined diagnosis as to the precise seat of the lesion there.

[1] Medical Times and Gazette, 1872, vol. I.

[2] Lehrbuch der Gehirnkrankheiten Bd. i, p. 353.

As regards loss of other associated motions of the eyeball, an interesting case of loss of motion of the eyes upward has been recorded by Gowers,[1] in which a small tumor was found
174, foot note.
in the middle line behind the posterior quadrigeminal bodies, damaging them slightly, as well as the velum and the adjacent part of the inferior vermiform process of the cerebellum. In a case of tubercle of the corpora quadrigemina, Henoch[2] observed this same defect as the first focal symptom to make its appearance.

Paralysis of the upward and downward motions of both eyeballs, sometimes with ptosis, while the lateral motions are unimpaired, may be the result of a focal lesion involving the third nerve nuclei in the floor of the Sylvian aqueduct; and, if attended by hemiplegia, the lesion involves the pyramidal tracts, probably at the level of the anterior quadrigeminal bodies, the posterior commissure, and the neighboring part of the optic thalamus. Lang and W. A. Fitzgerald reported a case to this society[3] in which this symptom and hemianopsia were the two focal signs. The case rapidly recovered, leaving only homonymous insular scotomata. In their communication the authors do not profess to have localised the lesion accurately, but I think this combination of symptoms might be accounted for by a lesion involving the pulvinar and anterior corpora quadrigemina.

Loss of the power of convergence, accompanied sometimes, as in Eales's interesting case,[4] by paralysis of accommodation, is a symptom of lesion of the posterior quadrigeminal bodies. Probably, however, this symptom is occasionally a distant one, for there are three cases published, one of them by Senator,[5] in which the lesion was in the pons, and did not involve the corpora quadrigemina.

[1]Medical Ophthalmoscopy, 2d Ed., p. 340, and Diseases of the Brain, 1888, p.
[2]Berl. Klin. Wochenschr., 1864, No. 13.
[3]Trans. Ophth. Soc., vol. ii, p. 230.
[4]Trans. Ophth. Soc, vol. iv., p. 300.
[5]Arch. f. Psychiatrie, Bd. xiv, p. 644.

A symptom which is the very opposite of conjugate deviation or paralysis may be best referred to here. It consists in a deviation of one eye downward and outward, while its fellow is turned upward and and inward. This remarkable, and, as yet, wholly inexplicable, symptom has only been seen with lesion of the middle cerebral peduncle, and the lesion may or may not involve the neighboring cerebellar substance. The last-mentioned symptom, as well as loss of power of the upward, or of the upward and downward motions of the eyeballs, are almost always direct symptoms.

We have now to consider the localizing value of those paralyses of the cranial nerves for the supply of the motor apparatus of the eye other than conjugate paralyses.

As regards the third nerve, we are at once struck with the fact that ptosis, partial or complete, may be present as a focal symptom in cortical lesions—cerebral ptosis, as it is called—without any other third nerve branch being paralyzed. That a separate cortical centre for this branch of the third nerve exists, and that it innervates the muscle of the opposite side, is very probable. The existence of such a centre would not be inconsistent with the view that, as regards the motions of the eyeballs, associated centres alone are present; for, although as a rule, the elevators of the lids are associated in their motions, yet by an effort of the will most people can throw one of them into motion separately, or more than the other. No doubt the power to voluntarily innervate one levator and orbicularis alone varies in different individuals, and Wernicke[1] leans to the opinion that in many persons the levator centres are practically associated centres, and that is the reason why cerebral ptosis is rather rare. The position of this centre is still an open question, for the view of Landouzy that it is situated in the posterior part of the inferior parietal lobule has not met with acceptance.

Ptosis, then, has no value as indicating the locality of a lesion in the cortex; but, according to Nothnagel,[2] it may be of

[1] Loc. cit., Bd. i, p. 323.

[2] Topische Diagnostik der Gehirnkrankheiten, p. 454.

use in distinguishing a cortical lesion from one situated elsewhere in the brain, for monolateral ptosis, as the only focal symptom, occurs with cortical lesions alone.

It is probable that ptosis, as the result of a cortical lesion, is a distant symptom in not a few of the cases where it is present.

Double ptosis was seen by Steffan[1] as the only focal symptom in a case of tubercular degeneration of the corpora quadrigemina, the neighboring parts of the brain being absolutely uninvolved in the lesion. It is interesting to note that in this case the motions of the eyeballs were not impeded, nor was there any defect of vision.

It is evident that lesions causing bilateral paralysis of branches of the third nerve which are wont to be innervated together—loss of motion of the eye upward, of motion of the eye downward, of convergence, and double ptosis—are to be sought for in the quadrigeminal bodies. Basal lesions do not give rise to similar paralysis.

Ptosis on the side of the lesion has occasionally formed a symptom in disease of the pons, without paralysis of the other branches of the third nerve—except, sometimes, in so far as conjugate deviation is concerned—and without the third nerve being involved in the lesion. An interesting case of this kind has been published by Wernicke.[2] Both upper lids drooped, the left more so than the right; there was also conjugate lateral deviation of the eyes to the right, the left eye being turned more inward than the right eye was turned outward. On an effort to look to the left the left eye did not move at all, while the right eye only reached the middle line. The whole of the left facial nerve was paralyzed, and sensation on the right side of the face and head was dull. These were only the focal symptoms, and they continued until the patient's death. At no time was there any paralysis, either of motion or of sensation, in the extremities. The carefully

[1]Berl. Klin. Wochenschr., 1864., Bd. vii, p. 513.

[2]Archiv f. Psychiatrie und Nervenkrankheiten, Bd. vii. p. 513.

made necropsy showed a tumor in the floor of the fourth ventricle, a little to the left of the middle line. Toward the left it did not extend beyond the lateral boundary of the pons; its posterior edge reached within $1^1/_2$ cm., of the calamus scriptorius, and its anterior edge was 1 cm., from the corpora quadrigemina, while the width of the tumor was 2 cm. The growth did not involve the middle peduncle of the cerebellum, but it did extend into the inferior cerebellar peduncle. The rest of the brain was normal. The zone of nerve tissue surrounding the tumor was free from all pathological changes, nor were there any traces of pressure in the pons or in the medulla. The upper half of the tumor was found to have involved, on the left side, the nucleus said to be common (Stilling) to the facial and sixth nerves. The nucleus of the small motor root of the fifth nerve on the same side had suffered, and the fibres of the descending root of the right fifth nerve had been interrupted. The lower part of the tumor had destroyed a large part of the facial nucleus, and a great part of the internal and external auditory nuclei, and the upper end of the glosso-pharyngeal nucleus. The nuclei and fibres of the third nerve were not in any way implicated in the lesion. It is as yet impossible to explain the occurrence of ptosis in this and other such cases—they have all been cases of tumor—unless we regard it as a distant symptom. It is important to note that in each of these cases paralysis of the facial was a constantly associated symptom. Consequently, in this combination, even if it should be a distant symptom, ptosis may have some localising value.

Again, ptosis, by forming a factor of a crossed paralysis, may serve to localise a lesion in the crus cerebri. When the third nerve is paralysed by a lesion in this situation, it is the rule to find it paralysed as a whole, but paralysis of only some of the third nerve branches may be produced by a lesion of the cerebral peduncle, and the branch to the levator palpebræ seems to be the one most frequently implicated alone. A case of this kind was published by Dr. Rickards,[1] in

[1]Journal, 1886, vol. i, p. 774.

which there was loss of motion and sensation of the left side of the body, with ptosis of the right eye ; later on, ptosis in the left eye appeared. The necropsy discovered a hæmorrhage of the size of a hazel nut occupying the upper and inner surface of the crura cerebri, but involving chiefly the right crus.

Another important case of this kind (important especially on account of the accurate necropsy) was published last year by Professor Leube.[1]

In order now to complete this subject of ptosis as a focal symptom, I must refer to a rare form of it which has been described by Nothnagel,[2] and which does not depend on a lesion of the third nerve. It may be called sympathetic, or pseudo-ptosis, and is accompanied by other eye symptoms as well as by symptoms of vasomotor paralysis of one side of the body, such as elevation of temperature and redness and œdema of the skin. In these cases, this author says, there is—1. Apparent ptosis on the paralyzed side, owing to the contraction of the palpebral aperture, but the lid can be raised. 2. Contraction of the pupil on the same side. 3. A shrinking back of the eyeball into the orbit, so that it seems to have become smaller. 4. An abnormal secretion of thin mucus from the corresponding nostril, of tears from the affected eye, and of saliva from the corresponding side of the mouth. Nothnagel states he had found this train of symptoms in lesions of the corpus striatum.

A common sign of disease of the crus cerebri is what is known as crossed hemiplegia. Paralysis of the third nerve on the side of the lesion, with hemiplegia, hemianæsthesia, often facial, and sometimes hypoglossal, paralysis of the opposite side of the body, is a frequent form of it. The lesion may implicate all the branches of the third nerve or only some of them. I have already given an instance in which only the branch to the levator palpebræ is involved. But the localising

[1] Deutches Archiv f. Klinische Medicin. Bd. xl, No. 2, p. 217.

[2] Loc. cit. p. 327.

value of crossed hemiplegia, as Hughlings Jackson long ago pointed out,[1] depends chiefly on the hemiplegia and paralysis of the cranial nerve coming on simultaneously. If they occur at different times, they may be due to two distinct lesions, neither of which may be in the crus; for the hemiplegia might be due to a lesion in the hemisphere, and the third nerve paralysis to a basal lesion of earlier or later date. Yet a few cases have been observed where, with a lesion in the cerebral peduncle, the third nerve paralysis preceded the hemiplegia by a considerable interval.

That basal lesions are by far the most frequent cause of paralysis of the third nerve is beyond a doubt; and here it is usual, but not constant, to find it paralyzed in all its branches. The diagnosis to be made, when direct symptoms are being considered, is, for the most part, between a lesion in the crus and a lesion at the base. We cannot pretend to be able to make this diagnosis with certainty in all cases. Complete paralysis of every branch of the third nerve without any other paralysis is almost always basal, so also, as Graefe pointed out, are those cases in which, where there is hemiplegia, it is slight as compared with the degree of the third nerve paralysis; and those cases, too, to which I have already referred, where there is an interval between the onset of the paralysis of the extremities and of the third nerve, are apt to be basal. Of course, there may be such a combination of paralysis of the other cerebral nerves with that of the third nerve as to leave no doubt with reference to the basal position of the lesion. But into all this I need not here enter.

Third nerve somytoms—in addition to those included under the headings conjugate deviation, or paralysis and ptosis—are sometimes distant symptoms. Tumors of the cerebral hemispheres, more particularly if accompanied by violent general head symptoms, indicating probably high intracranial pressure, are the lesions most apt to produce these distant third nerve symptoms. As a rule, the slighter the general cere-

[1]Lancet, Sept. 6, 1873.

bral symptoms are, the more likely are the third-nerve paralyses to be direct symptoms. This rule, indeed, applies to other, as well as to third-nerve, focal symptoms.

Paralysis of the fourth nerve, when combined with paralysis of other motor eye nerves, is difficult to recognize; and consequently, in such cases, it supplies but little aid for localisation. Solitary paralysis of this nerve, as a symptom of cerebral focal lesion, is extremely rare. Nieden has placed a case on record[1] in which paralysis of one fourth nerve was the only focal symptom to which a tumor of the pineal gland, of the size of a walnut, gave rise. But the isolated fourth-nerve paralysis is more apt to be produced by a basal lesion. In combination with paralysis of the third nerve it speaks for a lesion in the cerebral peduncle, extending back to the valve of Vieussens, and has, I believe, been utilised by Meynert in this sense.

Paralysis of the sixth nerve associated with paralysis of the internal rectus branch of the third nerve has been already considered. When paralysis of the sixth nerve occurs as the only focal sign, it is probably due to disease at the base, or it is a distant symptom. I think I am correct in stating that there is no cranial nerve so liable to provide a distant symptom as the sixth. Gowers refers this liability to the lengthened course these nerves take over the most prominent part of the pons, which renders them readily affected by distant pressure. One or both nerves may in this way be paralysed. I was recently consulted in a case in which there were diffuse cerebral symptoms, which left no doubt as to the presence of an intracranial tumor. The only focal symptoms were complete paralysis of the left external rectus muscle, and very slight loss of motion upward of the eyeball, and these had been present for only a few days. On one occasion, for a few minutes, there had been a tingling sensation in the left index finger. The shortness of the duration of the sixth-nerve paralysis rendered it, for the time, useless as a localising symptom

[1]Centralblatt f. Nervenheilkunde, 1879, No. 8.

and that it was really only a distant symptom seemed not unlikely from the fact that, as I was informed, there had been a few days previously paralysis of the right internal rectus, of which now there was no trace. Wernicke[1] states that sixth-nerve paralysis is most apt to be present as a distant symptom, when the lesion, especially a tumor, is situated in the cerebellum: differing in this way from the third nerve, which, as I have said, is more likely to give distant symptoms with a lesion in the cerebral hemisphere.

Paralysis of the sixth nerve, simultaneous in its onset with hemiplegia of the opposite side of the body, indicates a lesion in the pons, usually a hæmorrhage, on the side corresponding to the paralyzed nerve. We know that the fifth and facial, and sometimes the auditory, spinal accessory, and hypoglossal nerves, may all, in varying combinations, form one of the elements in a crossed paralysis from a lesion in this position; but, in the opinion of Nothnagel,[2] if special localising value is to be given here to the participation of any one cranial nerve, that nerve is the sixth. The paralysis of this nerve, simultaneously with palsy of the opposite side of the body, while other conditions point to an intracerebral lesion, speaks then almost certainly for pontine disease.

Paralysis of the facial with the sixth is not an uncommon combination caused by a lesion in the pons, which at the same time produces hemiplegia of the opposite side of the body. This combination is a natural one, in view of the close relations of the nuclei of the sixth and seventh nerves. Indeed, Lockhart Clarke, Meynert, and others, as you are aware, are of opinion that there is one nucleus which is common to both nerves, a view not shared in by Gowers and others. The manner in which the root of the facial nerve winds round the sixth-nerve nucleus must also have an important bearing on the occurrence of associated paralyses of these nerves.

Hemiplegia due to a lesion of the cortical motor region,

[1] Loc. cit. Bd. iii, p. 369.

[2] Loc. cit. p. 19.

which might happen to be combined with paralysis of the sixth nerve as a distant symptom, offers no difficulty in its diagnosis from hemiplegia with sixth-nerve paralysis in pontine disease; for while the latter is a crossed paralysis, the former is homonymous.

Lagophthalmos, we know, is the eye symptom to which paralysis of the facial nerve gives rise. It is useful for localisation, inasmuch as it assists in differentiating a lesion in the internal capsule, or in the facial motor centre of the cortex, from one implicating the portio dura in the pons, as it is absent or very slight in the former cases, but very often markedly present in the latter. With a lesion in the lower part of the pons we are apt to have lagophthalmos with crossed hemiplegia, but if the lesion be in the upper part of the pons, the fibres from the opposite side having here joined the motor tract, the hemiplegia and lagophthalmos will be homonymous.

Nystagmus, as a focal symptom, has little localising value. Indeed Nothnagel and Raehlmann agree in the view that it has no such value. It occurs as a distant symptom in many focal, as well as diffuse, cerebral diseases in various situations, but Gowers states that it is "especially common in tumors of the cerebellum."[1]

It will be convenient to refer here briefly to the eye symptoms of localising value afforded by paralysis of the fifth nerve. Similarly as the sixth nerve, crossed paralysis here points, to a lesion in the pons. Moreover, when the nucleus or fibres of the nerve in the pons are diseased, neuroparalytic ophthalmia rarely supervenes: while, if the lesion be basal, the corneal affection is the rule.

The condition of the pupils is rarely of much value in regional diagnosis. Bilateral myosis is often seen with hæmorrhage in the pons, and is usually reckoned amongst the more important symptoms of the lesion; but it is by no means a constant symptom of it, the pupils here being frequently of normal size. Although myosis is most commonly the result

[1]Loc. cit. p. 193.

of pontine lesions, yet it may accompany disease in other parts, as for instance, hæmorrhage in the corpus striatum, which bursts into the lateral ventricle, and in meningeal hæmorrhages. Bilateral mydriasis is frequently present in apoplectic coma, without reference to any particular locality. The same holds good as regards monolateral myosis and mydriasis.

To the monolateral mydriasis, which forms one of the symptoms of paralysis of the third nerve, I shall refer only to point out that this monolateral paralytic mydriasis has greatly assisted in localising a lesion in the cerebral peduncle. Rudolf Arndt has recorded a case[1] in which, among the early symptoms, the most marked were uncertainty and occasional loss of power of both lower extremities, incontinence of urine and of fæces, vomiting, and frequently recurring dilatation of the left pupil, no other branch of the third nerve being at the time affected. The necropsy discovered, as had been foreseen, a tumor in the intrapeduncular space, which pressed more on the left than upon the right peduncle.

Loss of the pupillary reflex to light, apart from cases of paralysis of the third nerve, is a sign of lesion of the anterior quadrigeminal, bodies, or of the optic tracts, and may be utilised to distinguish these lesions from others which may cause loss of sight by implicating both visual paths beyond the corpora quadrigemina, or both visual centres; for, in such cases, notwithstanding the amaurosis, the pupil reflex is maintained. Gudden's investigations[2] showed that there are special afferent fibres in the optic nerves and tracts for the pupil reflex, distinct from those for vision.

In case of hemianopsia, similarly, the pupil reflex serves to establish a diagnosis between a lesion in an optic tract and one further on in the visual path, or in the visual centre, of the same side; for, if the pupil contracts actively to light concentrated on the blind side of the field, the lesion cannot be in

[1]Archiv f. Psych., iv. 2. p. 432.

[2]Sitzungber. d. Muench. Ges. f. Morphol. u. Physiol. 1886. p. 168.

in the tract; but if it does not react, the lesion must be in the tract. It must be admitted that to decide the presence or absence of this hemiopic pupil is not always a simple matter, owing to the diffusion of the light when thrown into the eye, yet with care it may be obtained, or at least a marked difference in the promptness of reaction of the pupil, according as the light is concentrated more on the seeing or on the blind side of the retina.

And now, as to the second part of my subject—the localising symptoms derivable from the visual apparatus. Of these hemianopsia is one of the most common, as it is one of the most valuable. I may at once dismiss the three forms of hemianopsia which are known as temporal, nasal, and altitudinal, for they are only found with lesions of the chiasma, and provide a means, upon which I need not dilate, of localising those lesions at one or other side, above or below the chiasma. But complete and absolute homonymous lateral hemianopsia is often a symptom of great localising value. It may be caused by a lesion in the cerebral cortex, or by one situated anywhere in the course of the fibres between the cerebral cortex and the optic chiasma; and, by taking concomitant symptoms into account, we are frequently enabled to say in what part of this course the lesion lies.

By complete hemianopsia we, of course, mean, that the defect in the field extends up to the vertical dividing line, and by absolute hemianopsia, a defect which involves each of the three visual perceptions—color, light, and form.

And, first, as regards hemianopsia due to a lesion in the cerebral cortex, a lesion of the visual centre. Pathological anatomy leaves no doubt, but that, in man, the visual centre is situated in the occipital lobe rather than in the angular gyrus or elsewhere, and the evidence goes to show that the absolute optical centre chiefly occupies the cortex of the cuneus and of the superior occipital convolution, and also, especially in respect of the color sense, the posterior part of the superior and inferior occipito-temporal convolutions. The most important evidence to this effect is supplied by four cases pub-

lished respectively by Haab,[1] Huguenin,[2] Féré[3] and Seguin,[4] in which homonymous hemianopsia was present for a length of time before death, and in each of which the only cerebral disease consisted in a circumscribed lesion of the cuneus. Also an interesting case published by Bouveret,[5] in which sudden blindness of both eyes was caused by arterial thrombosis, resulting in softening of a symmetrical region on the mesial surface of each hemisphere, which included the cuneus on each side, and, it is true, a considerable extent of the occipito-temporal convolutions.

Then Berger, and Nothnagel[6] have each observed cases in which the lesion causing hemianopsia was restricted to the superior occipital convolution. Seguin, indeed, would confine the absolute optical centre to the cuneus, but Nothnagel seems to have good grounds for extending it over to the superior occipital convolution as well. Nothnagel points out that in nearly all the cases of extensive lesion of the occipital cortex which gave rise to hemianopsia, the cuneus and the superior occipital convolution were both implicated. He also draws attention to the fact that the middle and inferior occipital convolutions, the lingual and fusiform gyri, may all be disorganised without hemianopsia resulting. He is speaking here of absolute hemianopsia, but he does not discuss hemiachromatopsia, nor the question of separate centres for the three visual perceptions, and Verrey's case of hemiachromatopsia, to which I shall shortly again have to refer, makes it tolerably certain that the centre for the color sense is in the posterior part of the occipito-temporal convolutions.

Yet cases of hemianopsia are on record in which the disease

[1]Monatsbl. f. Augenhlk., 1882, p. 149.

[2]Monatsbl. f. Augenhlk., f. 1882, p. 143.

[3]Quoted by Seguin.

[4]Archives de Neuralgie, 1886, p. 176.

[5]Revue Generale d' Ophthalmologie, Nov. 1887. p. 481.

[1]Verhandlungen des VI. Congresses f. Innere Medicin, 1887.

has attacked one or other or all these parts, and has left the cuneus and superior occipital convolution unscathed. In some of these cases the lesion, no doubt, extended deep enough to involve the optic fibres on their way from the cuneus and superior occipital convolution; but in others it certainly did not do so. For these exceptional cases Nothnagel thinks that, as there is sometimes a departure from the usual anatomical arrangement of the convolutions of the occipital lobe, so, also, there may, in these cases, be an apparent irregularity in the distribution of its functions.

I may here mention that Reinhard[1] inclines to the view that at a point in the superior occipital convolution, close to the intra-parietal fissure, there is a half-vision centre for the macula lutea. Should this prove to be correct, a lesion of the cortical centre for vision which included this spot should always give a hemianopsia, of which the dividing line would pass through the fixation point. These observations of Reinhard's require confirmation, for his paper, being the product of investigations made upon persons of unsound mind, is necessarily unreliable in many particulars. It is especially so as regards the field of vision, for perimetric observations could not be made.

Wilbrand, also, believes that the macula lutea is specially represented in the visual centre.

But, as far as our present knowledge goes, it seems most probable that the view held by Gowers regarding the variations in the dividing line in hemianopic fields is the correct one, namely, that the slight differences with which we are familiar in the position of this line, with reference to the fixation point and to the central vertical line of the field, depend upon slight individual differences in the decussation of the central optic fibres, and have no important localising value.

It would appear, then, that we cannot expect to be able to distinguish clinically between an absolute hemianopsia due to a lesion confined to the absolute visual centre, with or without its efferent fibres, from one due to a lesion involving

[1]Zur Frage der Hiralocalisation, etc., Arch. f. Psych., Bd. xviii, II, p. 478.

at the same time other parts of the occipital cortex; and this we find to be borne out in practice.

But can we distinguish a complete and absolute hemianopsia, due to a lesion confined to the occipital lobe, from a similar defect in the field, due to a lesion in the optic radiations, internal capsule, pulvinar, or optic tract? We may conclude that the hemianopsia depends upon an occipital lesion, if it be unaccompanied by hemiplegia, motor aphasia, or paralysis of cerebral nerves, as direct symptoms—as might occur with a lesion in the posterior limb of the internal capsule on the left side (*vide infra*;)—but, be it remembered, that one and all of these are liable to accompany lesions of the occipital lobe as distant symptoms.

Aphasia, too, occasionally accompanies right cortical hemianopsia as a direct symptom. It is not easy to offer a satisfactory explanation of this fact, unless we accept Naunyn's view,[1] that there is sometimes a centre for speech in the region where the angular gyrus passes over to the occipital lobe, a region, consequently, very close to the centre for vision. It is also close to the centre for visual memory of words; and hence, aphasia, hemianopsia, and word-blindness—of which more later on—may be found in varying combinations in different cases. The form of aphasia, which Naunyn describes as produced by a lesion in this locality, is not altogether sensory, nor altogether motor, but is an undefined or mixed form. It is, moreover, often caused by a lesion in Broca's lobe, or by one in the centres which usually give sensory aphasia. But in about 40 per cent. of the cases, the lesion producing this undefined aphasia is, according to Naunyn, in neither of these localities, and then this part of the angular gyrus—that long-suffering region—is found to be the most common seat of disease.

Cortical hemianopsia may be a distant symptom. Gowers has observed that, at the onset of many attacks of cerebral hæmorrhage, hemianopsia is present at a distant symptom of very fleeting character, so fleeting indeed that it does not com-

[1]Verhandlungen des VI. Congresses f. Innere Medicin zu Wiesbaden, 1887.

plicate attempts at localisation. Except under this condition, distant hemianopsia seems to be rare, a fact which enhances the localising value of the symptom.

Cortical hemianopsia may be incomplete, but we do not as yet know that a lesion of the visual centre can be so situated as to produce loss of precisely the upper or lower half of the half field; yet the clinicists should bear in mind those interesting experiments made by Schäfer[1] on monkeys, which show that in these animals, as Munk had already proved for dogs, there is a correlation between the parts of the retina and of the occipital lobe. Removal of a certain part of each occipital lobe in the monkey gave rise to loss of the lower part of the field only in each eye. In future cases of incomplete cortical hemianopsia in which necropsies can be obtained, it will be important to note with the greatest possible exactness the precise extent and position of the disease.

So much for absolute hemianopsia. But the lesion may be such as to destroy only the color centre, without reaching those for form and light. Eight cases of hemiachromatopsia are on record.[2] There are also some cases of loss of color-vision in the whole field of each eye with retention of the form and light-senses.[3]

Again, the form-sense may be lost in the half field along with the color-sense, while only the light-sense is retained. Such cases are hardly less rare than the loss of the color-sense alone. Furthermore, cases of hemianopsia are on record[4] in which, in part of the defect, both the color and form-senses

[1]"Experiments on Special Sense Localisation in the Cortex Cerebri of the Monkey," Brain, Parts 39 and 40, January, 1888.

[2]1. Charpentier, Thèse de Paris, 1877 2. Bjerrum. Centralbl. f. Augenheilk., 1881, p. 471. 3. Samelsohn, Centralbl. f. d. Med. Wissensch., 1881. Nos. xlvii and l. 4. Swanzy, Trans. Ophth. Soc. vol. iii, p. 185, 1883. 5. Eperon, Arch. d' Ophthal., Juillet, 1884. 6. Willbrand, loc. cit. 7. Landolt, De la Cécité Verbale, 1888. 8. Verrey, Arch. d' Ophthal., Juillet, 1888.

[3]Wilbrand, Zur Diagnostik der Gehirnkrankheiten, 1884, p. 24; Steffan, Arch. Ophthal., xxvii, 2, p. 11.

[4]Wilbrand, loc. cit.

were absent, but the light-sense present, while in the remainder of the defect all three visual perceptions were lost. In some of these cases partial recovery took place by the light-and form-senses returning, while the color-sense still remained wanting; or, it may be, that the functions which returned had merely played the part of distant symptoms.

Until quite lately we had no necropsy in a case of loss of the color-vision alone. But Dr. Verrey, of Neufchâtel, has been so fortunate as to be able to publish, in the last number of the *Archives d' Ophtalmologie*,[1] a case which practically fulfils these conditions. Dr. Verrey is also to be congratulated upon the good sense which led him to submit his valuable preparation to the skilful handling of Dr. Burkhardt. The patient was a lady, 60 years of age, who consulted Dr. Verrey for a difficulty of sight she experienced in continued reading. This was the result of an apoplectic attack, from which she had rapidly recovered, without any other symptom of which she complained remaining. A perimetric examination showed that there was absolute color-blindness in the whole of the right side of the field of each eye, with diminution, but no absolute loss of the light-and form-senses in the same part of each field. Dr. Verrey considers that the patient's difficulty in reading was due to cerebral asthenopia, and expressly states that she had no dyslexia, properly so-called, nor any word-blindness. He also does not think that the slight diminution in the light-and form-senses in the right side of the field would account for the difficulty in reading which was present. In the course of the following twenty months, during which the patient lived and enjoyed fair general health, Dr. Verrey examined the fields several times, and found them unchanged. Finally the patient succumbed to another apoplectic attack.

The cause of death was found in a fresh hæmorrhage in the right centrum ovale and lateral ventricle; while an old hæmorrhagic cyst, in the lower part of the left occipital lobe, extending into the temporal lobe on the mesial side, explained the

[1] T. vii, No. 4, Juille-Août, 1888.

hemiachromatopsia. This cyst was situated between the floor of the posterior horn of the left lateral ventricle and the basal surface of the occipital lobe. It had occupied the white substance of the inferior occipital convolution, and had almost completely destroyed the white substance of the posterior extremity of the occipito-temporal convolutions, as well as that of the postero-inferior part of the cuneus. The cyst came almost to the surface of the cuneus and of the occipito-temporal convolutions, having destroyed the deeper layers of their cortex.

There can, then, be no doubt but that the centres for the color form, and light senses are all present in the occipital lobe and, posterior end of the occipito-temporal convolutions; and it is probable that they are either. as Wilbrand suggests, arranged in layers one over the other in the cortex, or, as others think, they are placed side by side. The latter is the arrangement Dr. Verrey deduces from his case, and he thinks the color-sense occupies the most inferior part of the occipital lobe, and probably the posterior part of the occipital lobe, and probably the posterior part of the lingual and fusiform convolutions of the temporal lobe; while higher, and more towards the superior part of the occipital lobe, is situated the cortical centre for the light-sense; and probably between these two is the centre for the form-sense, for the latter was the function which, after the color-sense, he found most affected. This agrees in the main with the view of Seguin and Nothnagel.

Relative hemianopsia can only occur with lesions of the cortex, hemianopsia from lesions elsewhere must always include all the visual perceptions. Those cases of hemianopsia, also, with some peripheral contraction of the other side of the field, are due to cortical lesions.

Hemianopsia from a lesion in the optic radiations will often be indistinguishable from the same symptom due to a cortical lesion. The defect may be incomplete, as the lesion may implicate only some of the radiating fibres; or it may be complete, if they are all involved. Pronounced distant symptoms, such as hemiplegia, hemianæsthesia, ptosis, and so on, are more apt to be caused by a lesion here than in the cortex.

A lesion in the posterior third of the posterior limb of the internal capsule—the sensory crossway—is likely to produce complete hemianopsia; because the nerve fibres are here collected together in a small space. Hemianæsthesia will be present as an accompanying direct symptom: and also, sometimes, loss of the other special senses on the opposite side from the lesion; and, should the disease extend forwards to the anterior part of the posterior limb, hemiplegia will be added as a direct symptom. Moreover, if the lesion be on the left side, motor aphasia may be present, by reason of the proximity of the path for speech on its way to the cerebral peduncle. I have already spoken of the combination of cortical hemianopsia with aphasia.

There are a few cases on record of hemianopsia caused by a lesion in the pulvinar. The symptoms in such cases strongly simulate those present in many cases of cortical hemianopsia, so that a differential localisation as regards these two positions may be impossible. The hemianopsia will be absolute, and probably complete; but lesions just in this situation seem to be very rare.

In hemianopsia due to a lesion of the optic tract the defect in the field is usually complete. The characteristic sign which enables us to localise a lesion in this position from one elsewhere causing hemianopsia is the hemiopic pupil already described.

Two cases may be mentioned which exemplify the peculiar service this pupil-symptom is sometimes capable of rendering. One of these—interesting, too, for other reasons—is published by Brandenburg in the last volume of Graefe's *Archiv*.[1] In the right eye there was a hemiopic defect of the field to the right side. But the field in the left eye could not be examined, owing to complete leucoma of the cornea. The defect in the right field, therefore, might have been the representative of a bitemporal hemianopsia due to a lesion at the chiasma, in so far as the perimeter could decide. The absence of the hemi-

[1] Bd. xxxiii, 3, p. 93.

opic pupil it was which showed that the lesion was not at the chiasma, and, no doubt, if the field of the left eye could have been examined, a homonymous defect would have been found in it.

The other case, observed by Brieger, is quoted by Wernicke.[1] In it there was complete blindness of both eyes, and, as the pupil in a general way reacted to light, a diagnosis of double hemianopsia due to lesions of both occipital lobes was made. At the necropsy only the right occipital lobe was found diseased, while the left occipital lobe was healthy; consequently, the left-sided hemianopsia alone was so far accounted for. But the cause of the right-sided hemianopsia was soon discovered. In consisted in a degeneration of the left optic tract. If, then, during life the pupillary reaction had been more carefully examined, the existence of two lesions, one in the left tract and the other on the right side, beyond or higher than the tract of that side, could have been diagnosed; for the pupillary reflex would have been found wanting or defective if light had been concentrated on the left half of one or other retina.

Lesions of the optic tract are, of course apt to implicate the crus cerebri, but do not necessarily do so. Leber has pointed out that atrophy of the optic nerve is likely to make its appearance and at an early stage of the case, in lesions of the tract.

Total blindness of both eyes, when it appears as a focal symptom—apart from such cases of double cortical hemianopsia, or of cortical hemianopsia combined with a lesion of the opposite optic tract, as I have already referred to—can only be due to a lesion involving the whole of the chiasma, or of both optic tracts. The great mass of clinical evidence is opposed to the idea that lesions of the corpora quadrigemina produce blindness.

I must now ask you to return with me for a moment to the occipital lobe. Visual auræ have sometimes been noted as a

[1]Loc. cit., Bd. iii, p. 335.

symptom of disease of this lobe. Gowers has recorded a case[1] in which flashes of light were seen before both eyes, but especially before the left eye, and in which the necropsy discovered a sarcomatous tumor occupying the first and second occipital convolutions, the posterior half of the superior and inferior parietal lobes, and, on the mesial surface, the cuneus and the præcuneus. Other authors, too, have recorded occipital tumors with similar symptoms.

A very remarkable visual defect is that known as mind-blindness, or loss of visual memory—blindness, we may say, of what has long been known as "the mind's eye." Sight in the ordinary sense of the word—the reception of the retinal images by the visual centre—is unimpaired, but the psychical realisation of the retinal images is not effected. The objects are seen, but the sight of them suggests no corresponding idea in the patient's mind.

We are acquainted with several varieties or degrees of this symptom. In a well-marked case the patient may be unable to recognise the streets of the city in which he has been resident for many years, and will feel as if in a strange place; he may not know his own hall door. He may be unable to distinguish his wife from his mother, and his children may appear as strangers to him. He will be unable, when away from them, to recall to his mind's eye the appearance of the places and people that have been familiar to him all his life. Yet such a man will be perfectly capable, so far as his other intellectual faculties are concerned, of transacting business, and of entering into the enjoyments of life.

In a case which I brought before this Society five years ago; mind-blindness of a partial character was one of the symptoms, although at the time I did not sufficiently direct attention to it; for I did not myself understand its significance. The case is recorded in our *Transactions*[2] as one of hemiachromatopsia, but it was partial mind-blindness which brought the gentle-

[1]Lancet, 1879, Vol. 1, p. 363.

[2]Loc. cit.

man, aged 77, to me. Five months previously he had had a slight apoplectic attack, and he came complaining that ever since then he could not see people properly, although he saw everything else as plainly as ever. Then he expressed himself differently, and said he did see people indeed, but did not know them, even when near to them. His own wife he would pass in the street, and if she stopped him, he would look straight in her face, she told me, without any expression of recognition in his until she spoke, and he then at once knew who it was. He said that although he saw me at that moment quite plainly, yet if he met me unexpectedly five minutes later, my appearance would be as new to him as though he had never seen me before; but he always identified people when they spoke. He was hypermetropic 1.5 dioptries, and had an acuteness of vision of $^{6}/_{XII}$. With the exception of some peripheral cataract, his eyes were organically sound. In the course of the functional examination I discovered the hemiachromatopsia, which afterwards formed the main subject of my communication, although in my paper I did mention his difficulty of recognising people, and stated that I regarded it as a disturbance of a cerebral function. In addition to the difficulty of recognising well-known faces, the patient only complained of a confusion of ideas, when he made an unwonted mental effort. He did not tell me anything about color-blindness, until he saw that I was going to test the color-sense, and then he said he had found it most difficult to distinguish colors since his attack.

Mind-blindness is seen in cases of general paralysis, usually in the advanced stages. In one case under Wernicke's care[1], and which was examined by Schweigger—where the symptoms came on at an early stage, the patient's intelligence being still good—the remarkable circumstance was noted that, with good acuteness of vision, and without any absolute defect in the field, there were, disseminated over a great portion of the field, a number of relative scotomata, within the area of any

[1]Loc. cit., Bd. iii, p. 552.

one of which, although objects could be seen by the patient, yet he could not recognize them, could not tell what they were. The same objects in other parts of the field he was able, not only to see, but also to recognize. The region of the macula lutea did not correspond to one of these scotomata. Wernicke regrets that the color-vision in the scotomata was not tested, and it would, indeed, have been interesting to know what its condition there may have been.

The position of the cortical centre for visual memory is still a subject of discussion. Hitherto all the necropsies have been made in cases of general paralysis, and for definite knowledge we must wait for the post-mortem examination of a case in which the symptom has been caused by cerebral hæmorrhage. Nothnagel, Wernicke, Wilbrand, and some other writers assign this function to all, or to most, of that part of the occipital cortex which does not form the centre of vision. Gowers thinks it is either in the anterior part of the occipital lobes, or in the posterior part of the parietal lobes; but the latter, he believes, is the more probable. The curious and interesting fact that, in Charcot's case, in a case recorded by Quaglino, in one by Landolt, and in my own case, a derangement of the color-sense came on simultaneously with mind-blindness seems strong evidence in favor of a localisation of visual memory very close to the visual centre. My case is, perhaps, of special importance in this connection, as the lesion was evidently of slight extent. Cases, too, are on record in which absolute hemianopsia accompanied mind-blindness. I should, therefore, be inclined, in the absence of a conclusive necropsy, to localize this function very close to the centre for vision, probably in the occipito-temporal lobe.

Gowers thinks that when mind-blindness results from disease of one hemisphere, it is probably transient; but my case, where the lesion was evidently in the right hemisphere, and where, even when I first saw the patient, the symptoms had lasted five months, does not support this view.

It would seem that mind-blindness may be a distant symptom, for Wilbrand records a case in which it appeared to have only this significance.

I must here refer to a case reported by Dr. Macewen, of Glasgow, in his remarkable address on "The Surgery of the Brain and Spinal Cord," delivered at the last meeting of the British Medical Association.[1] A man, who had received an injury about a year previously, suffered from deep melancholy and strong homicidal impulses, which were relieved by paroxysms of pain in the head of indefinite seat. The only localising symptom Dr. Macewen could obtain was mind-blindness, which had been present immediately after the accident, and for about two weeks subsequently. On operation, the angular gyrus was exposed, and it was found that a portion of the internal table of the skull had been detached from the outer, and had exercised pressure on the posterior portion of the supra-marginal convolution, while a corner of it had penetrated and lay imbedded in the anterior portion of the angular gyrus. The bone was removed from the brain and reimplanted in proper position, after which the patient became greatly relieved in his mental state, though still excitable.

Dr. Macewen thinks that the definite localization in this case will assist in indicating, in man, what function the anterior portion of the angular gyrus and the posterior portion of the supra-marginal convolution subserve. The brilliant result obtained places the case almost beyond the region of any criticism; and yet I cannot help expressing the view that Dr. Macewen ventured a good deal here in adopting the mind-blindness as a localising symptom. The fact that it was present immediately after the accident for only a fortnight, although in marked degree, would raise a strong suspicion that the mind-blindness may have been a distant symptom, and nothing more. Again, I do not quite agree with Dr. Macewen in thinking his case conclusive as to the function of the injured convolutions. If visual memory be their function, why was it so completely destroyed in the first fortnight during which the lesion existed, and completely restored from that time on, although the lesion remained for a year afterwards? The cen-

[1] Journal, Aug. 11, 1888.

tre for visual memory may, indeed, reside in the angular gyrus; but I hardly think Dr. Macewen's interesting case can be taken as a proof that it does reside there.

Word-blindness, or alexia—loss of the power of understanding printed or written speech-symbols—is held by many to be nothing more than partial mind-blindness. Gowers, Wernicke and Wilbrand are of this opinion, and all these authors, as well as Ferrier and Broadbent, localize the lesions which produce the symptom in the angular gyrus of the left hemisphere. Nothnagel,[1] while he localises the centre for visual speech-symbols in this same region, dissents from the view that word-blindness is to be included in mind-blindness; and the fact that in some well-marked cases of mind-blindness there was no word-blindness might seem to support this view. But, if the centre for visual memory of every kind, except that for printed and written speech symbols, is situated in the occipital lobe, as Nothnagel himself holds, there is no reason why word-blindness should accompany mind-blindness, unless in those cases in which the lesion extends to the angular gyrus. The fact that word-blindness may exist without mind-blindness is, for a similar reason, no argument in favor of Nothnagel's opinion.

A good many cases of alexia with right hemianopsia have been recorded, and a natural explanation of this combination of symptoms is supplied by the proximity of the centre for vision to the angular gyrus. Indeed, some authors go so far as to state that hemianopsia is present in all cases of word-blindness. It is interesting to note that there is one case, reported by Dr. Borel,[2] in which, in a right-handed person, word-blindness along with mind-blindness accompanied left hemianopsia. And Landolt[3] has placed on record a case of right hemiachromatopsia in which there was word-blindness.

I may be permitted to briefly refer to a peculiar variety of

[1]Verhandlungen des VI. Congresses f. Innere Medicin zu Wiesbaden, 1887.

[2]Landolt, loc. cit.

alexia which has been seen in two cases, although we cannot at present utilize it for the purpose of localisation. One of these is reported by Brandenburg[1]. The patient, who was also affected with right homonymous hemianopsia, was unable to read any printed or written letters or words, yet he could read off with ease long numbers reaching to tens of thousands in Arabic characters. In the second case—quoted by Brandenburg—Joly[2] observed the same power of reading Arabic numbers, while the power of reading words and letters was lost.

I do not know that alexia has been seen as a distant symptom.

The remarkable symptom termed dyslexia was first described by Berlin.[3] He has observed it in six cases, and Nieden[4] and Bruns[5] have each recorded one case of it. The patient is unable to read more than a very few words consecutively, either aloud or to himself, owing to a feeling of dislike or disgust which suddenly invades him, and which he cannot overcome. After he has read a few words, which he can understand well, he pushes the book away or hands it to the surgeon, while he draws his head back, and turns it aside. After a brief interval the attempt may be renewed, but with the same result, when a few words have been read. There is no dimness of sight, defect of accommodation, or pain in the eyes or head to account for the symptom. It usually comes on suddenly, and is the first sign of serious cerebral disease, being soon followed by other symptoms, such as headache, giddiness, aphasia, hemianopsia, hemiplegia and so on. All of these cases have ended fatally. The details of the necropsies, in those cases where they were obtained, leave much to be desired. In every instance the lesion was on the left side of

[1]Loc. cit:

[2]Amnesie et Cécité verbale, Le Scalpel, 1883, No. 24, p. 147.

[3]Arch. f. Psych., Bd. xv., p. 276, and in his monograph: Eine besondere Art der Wortblindheit, Dyslexia, Wiesbaden, 1887.

[4]Arch. f. Augenhlk., Bd. xviii., p. 162.

[5]Neurolog. Centralbl., Nos. 2 and 3, 1888.

the brain, the patients being all right-handed. The disease seems to have occupied chiefly the inferior parietal lobule, extending sometimes as far forward as the inferior frontal convolution, and sometimes as far backward as the angular gyrus. It is evident, however, that we must be content to wait for more definite information with regard to the usual seat of the lesion, before this symptom can be utilized in practice for the purposes of localisation.

In crossed amblyopia—in which the eye on the side away from the cerebral lesion is almost blind, with very contracted field, while the field of the other eye is also contracted, but in a less degree—the lesion has been found in the lower and hinder part of the inferior parietal lobule.

I must mention that so distinguished an author as Nothnagel, writing about a year ago,[1] expressed his doubt as to the occurrence of crossed amblyopia. He does not think the perimetric examinations have been made in these cases with care sufficient to exclude the possibility of their being examples of incomplete hemianopsia. Nothnagel does not offer any experience of his own, and I am slow to think that observers like Ferrier, Gowers and Sharkey could fall into such error.

In conclusion, with reference to optic neuritis, it is merely necessary to state that it has practically no localizing value; nor should I think it is necessary even to do this much but that, in some recent cases of brain surgery, it seems to have been tried to utilize the optic neuritis for localisation. Optic neuritis occurs in most cases of intra-cerebral tumor, irrespective of the seat of the disease.

And now, gentlemen, I have come to the end of this lecture, and have not as yet made any reference to that distinguished man in whose honor I have the honor to address you. But I do not believe you could wish that we should separate this evening without your having heard from me some expression of the esteem in which we hold Sir William Bowman, nor will I be guilty of such an omission. And here I might dwell upon

[1]Congress fuer Innere Medicin, Wiesbaden, 1887.

Bowman's scientific attainments, upon the good work he did in years gone by—not alone in ophthalmology, but also in physiology—upon all this Society owes to his prestige and to his generosity, upon his clinical knowledge and operative skill, and upon many another topic which his name suggests. But I prefer to remind you of the high standard of professional life he has shown us, a standard which we of a younger generation must endeavor to maintain. Conscientious in his relations with his patients, honorable in his relations with his professional brethren, careful not to put himself forward in any unrecognized manner, not seeking notoriety, simple, kind, courteous, dignified, William Bowman is presented to our mind's eye as the personification of the best of those qualities which go to make an English gentleman. We are indeed privileged in being permitted during his lifetime to offer some tribute to his distinguished scientific merit; but, while recognizing how much we owe him scientifically, I think, and I believe you will admit, we should not forget how much his everyday professional life has tended to give to the noble specialty he adorns that high tone which belongs to it. Our sincere hope is that Sir William Bowman may long live to enjoy the honor conferred on him by his Queen, the love of those who are dearest to him, and the warm, heart-felt esteem of this Society.—*British Medical Journal*